Before, after, & after-after:

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Clinical implications of weight loss recidivism

Abstract: Weight recovery among obese patients who have lost weight through lifestyle modification or bariatric surgery is a common clinical challenge that often leads to patient stigmatization and unexpected health problems. A review of the literature describes how weight loss alters energy homeostasis to limit weight loss and restore lost fat mass in patients who have successfully lost weight.

By Mary Madeline Rogge, PhD, RN, FNP-BC and Bibha Gautam, PhD, RN, CNE

Obesity is a well-recognized global health problem. Worldwide, more than 600 million adults have a body mass index equal to or greater than 30 kg/m^2, and more than 42 million children under age 5 are overweight or obese.[1] Obesity increases the risk of these individuals to develop chronic comorbidities, including cardiovascular diseases, cancer, depression, insulin resistance, and type 2 diabetes mellitus. Based on anecdotal evidence, obesity also takes a physical and emotional toll on the individuals it affects through their efforts to lose weight, only to regain the weight they fought so hard to lose.[2]

Many believe that individuals should be able to consciously and voluntarily determine their weight and fat mass by controlling the quantity and quality of food they eat and the amount of exercise they perform. According to this paradigm, to correct their excess adiposity, all obese individuals have to do is eat less and exercise more. This regimen creates a negative energy balance in which the body is forced to consume its stored fat for energy, and weight loss occurs.[3]

Healthcare providers prescribe various dietary regimens for weight loss: low-fat, reduced-energy diets; low carbohydrate, reduced-energy diets; Mediterranean-style diets; and low-calorie diets.[4] Interventions to reduce weight also include increasing exercise (especially aerobic exercise), behavior modification, pharmacotherapy, and bariatric surgery.[4]

Implicit in the calories in/calories out paradigm is the idea that once the obese individual adopts the prescribed lifestyle changes, these changes should become permanent habits, leading to optimal well-being at a new "healthy" weight.[5] This assumption has led to the common depiction of obesity management as "before and after" treatment. Most research and commercial advertisements regarding the benefits of weight loss regimens and products measure two points in time: before starting a treatment regimen and a few weeks or months after weight loss.[6,7]

However, there is a third point, which, until recently, has gone largely overlooked by healthcare professionals as well as commercial weight loss advertisers: the "after-after" phase. Research has shown that a wide array of treatment regimens can result in weight loss for a few months, but very few patients can sustain the weight loss permanently, regardless of the program's specific content.[8]

Similarly, the rate of weight loss (rapid versus gradual) does not affect the proportion of weight regained.[9] Treating

Keywords: obesity, obesity relapse, weight loss recidivism, weight recovery

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obesity is less about losing weight than it is about keeping weight off. One of the most important things NPs can do in caring for obese patients is help them understand the problem of weight regain after successful weight loss.

Weight regulation

Energy balance, weight, and adiposity are biologically regulated, and this regulation is under the control of a growing list of chemical mediators. NPs should be able to summarize major physiologic adaptations to caloric restriction, negative energy balance, and weight loss for their patients. They should also be able to explain how biologic adaptations to voluntary weight loss conserve energy expenditure, reverse restricted food intake, and restore adipose tissue.

Two groups of hypothalamic neurons regulate food intake, energy expenditure, and body weight; they receive signals from outside the hypothalamus and send signals to other regions of the brain and other effector organs, such as the thyroid gland and skeletal muscles, to coordinate energy balance. Orexigenic neurons and their neurotransmitters facilitate weight gain and fat accumulation by increasing appetite and food-seeking behavior. They also conserve the body's fuel stores by curtailing unnecessary energy expenditure, especially through unconscious and subconscious movement.[10] Major orexigenic molecules in the hypothalamus, which stimulate food-seeking behavior and diminish energy expenditure, include neuropeptide Y (NPY) and agouti-related protein (AgRP).

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These molecules promote hyperphagia and depress energy expenditure.[11,12] NPY also favors consumption of fuel-dense, high-fat/high-carbohydrate foods and stimulates the proliferation of adipocytes from stem cells and preadipocytes.[12,13] Ghrelin is an orexigenic mediator secreted by the stomach and is believed to initiate feeding behavior. One of the ways ghrelin promotes food consumption is by stimulating secretion of NPY and AgRP.[14,15]

Adenosine monophosphate kinase (AMPK) is produced when the rate of adenosine triphosphate (ATP) production falls below the rate of cellular energy utilization, as in intense or prolonged physical activity. Within the hypothalamus, AMPK augments the secretion of NPY and AgRP to stimulate hunger and reduce energy expenditure.[16,17]

In regulating energy balance and body adiposity, orexigenic signals are opposed by neurons producing anorexigenic peptides that promote weight loss by increasing satiety and lowering food consumption. The anorexigenic mediators also increase energy expenditure in part by raising the basal metabolic rate and increasing spontaneous nonexercise movement, such as changing posture, pacing, and fidgeting. Key anorexigenic molecules include leptin, glucose, and insulin.[15,18]

Normally, leptin is secreted by adipose tissue and acts in multiple ways to decrease feeding and increase energy expenditure.[12,15,19] Leptin inhibits NPY and AgRP secretion, thereby decreasing hunger and food intake. Leptin also promotes energy expenditure, in part, by activating the sympathetic nervous system (SNS). SNS activation increases lipolysis, the breakdown of lipids in adipose tissue, and liberates fatty acids to fuel cells.

Activation of the SNS further increases energy expenditure by increasing skeletal muscle tone and promoting glucose and fatty acid oxidation and by increasing heat production in brown fat. For reasons yet unknown, obese individuals have abnormally high leptin levels due to their high fat mass, but they are resistant to the anorexigenic action of leptin the way some individuals are resistant to the action of insulin.

Glucose is a powerful anorexigenic signal in energy regulation. Although healthcare professionals often classify sugars as "empty calories," they are not. Glucose is the primary source of energy for neurons.[20] The availability of glucose within the hypothalamus signals to the brain that fuel is available to meet the energy needs of cells (especially neurons) and reduces hunger and feeding behavior.[21,22] Conversely, declining glucose availability and utilization by hypothalamic neurons stimulates increased hunger and food-seeking behavior independent of the absolute glucose level.[22] Similarly, insulin is an anorexigenic signal that is thought to participate in energy regulation by inhibiting NPY/AgRP production. Increased hypothalamic insulin produces decreased food ingestion and greater energy expenditure. Secreted when blood glucose levels start to rise, insulin augments the anorexigenic or appetite suppressing message produced by glucose.[19]

Signals from the orexigenic and anorexigenic mediators are carefully choreographed to work together to maintain a stable energy balance, body weight, and adipose stores. A few hours after a meal, glucose, insulin, and leptin levels decline, whereas NPY and AgRP neurotransmitters rise to stimulate hunger, feeding behavior, and energy conservation.[18]

With less fuel available, concentrations of AMPK increase and augment the NPY and AgRP signals.[16] Surges of

ghrelin intensify the orexigenic signaling, so that at intervals, the individual is prompted to eat again to replenish available fuel for several more hours.[14]

Consuming food activates anorexigenic signaling and decreases orexigenic messenger molecules. Anorexigenic signals from glucose, insulin, and leptin increase, whereas secretion of NPY and AgRP declines. Feeding subsides and energy expenditure increases; metabolic energy expenditure and spontaneous physical activity also increase. These cycles of alternating secretion of anorexigenic and orexigenic chemicals maintain energy balance, body weight, and fat mass over the long term.[15,18] (See *Effects of diet, exercise, and weight loss*.)

Effects of diet, exercise, and weight loss[37,42]

When patients follow clinical advice to decrease their food intake to lose weight and adipose mass, their anorexigenic signals become depressed, and orexigenic signals are activated. With food restriction and weight loss, glucose, insulin, and leptin diminish, while ghrelin, NPY, AgRP, and AMPK increase. Likewise, when obese patients increase their energy expenditure through exercise, they do not just burn calories; they also reduce glucose and insulin concentrations and increase AMPK production.

Lower glucose and insulin, along with higher AMPK, will amplify the release of NPY and AgRP to restore energy homeostasis.[9] Obese individuals may attempt to increase their physical activity by 30 to 60 minutes daily as recommended for weight loss, but the body will adapt to increase food consumption to cover the increased energy expenditure. The body may also reduce other activities requiring ATP and increase skeletal muscle efficiency.

Systemic metabolic changes defending adiposity

Systemic physiologic changes occur as a result of the alterations in signaling molecules following reduced food consumption and weight loss. A reduction in weight reduces 24-hour energy expenditure by about 15%, which facilitates a return to the individual's baseline weight.[23,24] Both resting energy expenditure and physical activity energy expenditure decrease.[23-25]

The reduction in energy expenditure is accomplished by lowering energy spent in digestion (decreased thermic effect of feeding) and decreasing SNS tone while increasing parasympathetic tone. Triiodothyronine secretion is also reduced from baseline, although not necessarily depressed to clinical hypothyroidism.[23,24] Reduction in thyroid hormone mediates the decline in metabolism and energy expenditure associated with weight loss.

Energy conservation also occurs by decreasing the conversion of fatty acids to ATP, especially in muscle cells. Normally, most of the ATP produced daily is through the oxidation of fatty acids, and a weight loss of approximately 10 kg results in reduction of fat oxidation by approximately 20 g/day.[26] A reduction in fat oxidation by 20 g/day reduces energy expenditure by approximately 180 calories a day. It becomes harder for the weight-reduced obese individual to produce ATP and favors more sedentary behavior.[24,25]

The reduction in fatty acid oxidation makes it harder to expend energy through physical activity to continue losing weight. Clinically, individuals attempting to lose weight experience a plateau or begin to recover the adipose tissue they have lost as a means to restore energy balance.

It cannot be emphasized enough that the individual who has lost weight experiences a significant *involuntary* reduction in 24-hour energy expenditure. The individual who has lost weight must continue to eat 15% fewer calories to maintain the same weight as an individual with an identical body composition who has never been obese.[24,27] The decreased energy expenditure in the weight-reduced obese individual as well as altered levels of circulating appetite mediators has shown to persist for years or until the weight is regained.[24,27,28]

These metabolic changes are outside the intentional control of the individual attempting to manage obesity through diet, exercise, and behavior modification. The metabolic compensation for weight loss is achieved by altering energy expenditure, and most of this effect is achieved by changes in the amount of energy spent in activities of daily living, changing positions, and fidgeting rather than through changes in resting energy expenditure.[24,27]

Skeletal muscle efficiency

Skeletal muscle performance adapts to resist weight and adipose tissue loss. During maintenance of reduced body weight, adults demonstrate increased skeletal muscle efficiency. Fewer calories are expended per unit of work in weight-reduced obese adults.[25,29] The decline in energy expenditure is greater than the reduction in energy expenditure expected based on changes in body weight and composition.

Researchers have found that increased skeletal muscle efficiency is most pronounced in low-intensity physical activity but remains comparable to the nonreduced state at high exercise workloads.[29] The obese individual who adds 30 to 60 minutes of daily moderate or high-intensity exercise to facilitate weight loss offsets this voluntary energy expenditure by involuntarily increased muscle efficiency while performing other routine low-intensity activities the rest of the day.

The increased energy efficiency will oppose weight loss and help conserve fat mass. These findings do not mean obese individuals cannot benefit from exercise, but that

increasing exercise is not a panacea for individuals trying to control their weight.[30]

Psychological changes in weight loss

Weight loss usually has psychological and social effects in addition to physical effects. Researchers have demonstrated that restricting caloric intake increases total cortisol secretion, an indicator of increased physiologic stress, while monitoring caloric intake results in perceived (psychological) stress.[31] Changes in other neuropeptides, including dopamine and serotonin, will also affect mood. While trying to restrain food intake and maintain weight loss, the reduced-weight obese individual typically experiences increased fatigue, hunger, anger, and irritability; sense of cold; and a preoccupation with food.[32-35]

After weight reduction, homeostatic signals will drive a restoration of weight and fat mass through increased feeding and lower activity levels.

"Catch-up" fat

After weight reduction, homeostatic signals will drive a restoration of weight and fat mass through increased feeding and lower activity levels. When refeeding occurs and weight is regained, fat is recovered at a disproportionately faster rate than lean muscle tissue, even when the individual is consuming a diet relatively low in fat with an adequate protein content.[36,37]

"Catch-up" fat, or adipose recovery, is associated with reduced blood flow to muscles, increased free fatty acids, and elevated SNS activity. Unfortunately, the processes supporting catch-up fat increase the risk of hypertension, insulin resistance, and dyslipidemia.[37] A study of weight loss among postmenopausal women demonstrated that even partial weight recovery after a single cycle of weight loss was associated with a worsening metabolic and lipid profile.[37,38]

Adipose tissue can expand in two ways. One way to increase the fat mass is through adipocyte hypertrophy, where the adipocytes become larger and hold more lipids. The other way is through hyperplasia, where the number of adipocytes increases through the differentiation of stem cells to preadipocytes and adipocytes. Obese individuals generally have greater numbers of adipocytes and larger adipocytes than those who are normal weight.[27]

Weight loss and recovery affect adipocyte number and volume as well as lipid content and leptin secretion. Diet-restricted weight loss is associated with a reduction in adipocyte size and lipid content but not a reduction in adipocyte number. As a result, the adipose tissue's capacity to store lipids remains the same as before weight loss, but stored lipids fall well below capacity.[39] Smaller adipocytes become more responsive to insulin to clear glucose from the circulation and stimulate lipid formation. The smaller adipocytes are also more resistant to lipolysis, which is the breakdown of lipids for energy.

Thus, weight-reduced obese individuals often have improved blood glucose levels and lipid profiles early in weight reduction, but the smaller adipocytes are primed to replenish lipid stores at the same time involuntary neuroendocrine signals are spurring the individual to increase feeding. Furthermore, researchers have found that adipocyte hyperplasia occurs early in the refeeding process so that new, very small adipocytes are recruited and augment the fat mass in human adipose tissue recovery.[39] These new adipocytes are capable of hypertrophy and continued lipid accumulation in the postreduced state of obesity.

Obesity as a disease

In 2004, Medicare reversed a long-standing policy that obesity is not a disease. This acknowledged the rising research regarding the difficulty of weight control and paved the way for reimbursement of health services for obesity management, including nutritional, behavioral, and psychological counseling; diet programs; and bariatric surgery.[40] Because many private insurance companies base their covered services on Medicare policy, the change in policy paved the way for insurance companies to follow suit and classify obesity as a disease.

In 2013, two large health profession organizations recognized obesity as a disease, not just a behavioral defect. At its annual meeting, the American Medical Association (AMA) adopted a policy recognizing obesity as a disease to advance obesity treatment and prevention.[41]

Similarly, in January 2013, a panel of NPs met to discuss the problem of obesity and the role of NPs in obesity management. In the white paper they produced, the American Nurse Practitioner Foundation (ANPF) panel identified obesity as a chronic disease.[42] The white paper also noted NPs need to improve interventions and outcomes for factors contributing to obesity, such as lack of sleep and medications prescribed for other conditions. They also advocated for more obesity-focused educational and clinical resources for NPs in clinical settings.

While both the ANPF and AMA efforts are commendable for the stand they take to improve the care of obese patients,

they continue to focus on negative energy balance and weight loss as the way forward in obesity treatment. The problem with this approach is that the metabolic adaptation to obesity makes it unlikely the weight-reduced obese individual will be able to maintain the lower body weight over time. The expectation that the obese individual will sustain weight loss in opposition to the resulting metabolic adaptation will foster blaming the obese individual for "nonadherence" and stigmatizing their failure as a lack of willpower, self-discipline, or motivation.

Weight loss can provide temporary improvement in serum glucose and lipid levels, but these benefits are rarely sustainable. Furthermore, weight loss and adipose recovery processes carry other costs, including increased metabolic efficiency promoting greater fat recovery and loss of self-esteem associated with weight regain, which are disregarded in the assumption that weight loss will be maintained.

The current state of the science regarding obesity reveals that the calorie-based approach to obesity management is not effective over the long term and explains why diet and exercise fail to sustain successful weight loss. Existing knowledge does not offer overt solutions about how to enable the obese individual to lose weight and sustain weight loss. In an applied science such as nursing where NPs often expect to implement scientific solutions to clinical problems, the absence of a clear protocol to achieve sustained weight loss can be disheartening. However, the first step in expanding the scientific basis of practice is to point out the knowledge base limitations, reframe the problem, and envision new approaches to the problem of obesity.

The ANPF white paper is an important development in changing clinical care for obese patients because it acknowledges the need to individualize care and address "noncaloric" factors contributing to obesity, such as medications and lack of sleep.[42] NPs also need to research and adopt new treatment modalities to help patients cope with obesity and optimally live their lives in an obese state the way NPs adapt care for patients with recurrent cancer or progressive neurologic dysfunction until new solutions are available. Educating obese patients is a priority service that NPs can provide detailing why they relapse when they work so hard to manage their weight.

NPs managing obesity care

As clinicians in the forefront of identifying and managing obesity, NPs must take a leading role in educating colleagues, patients, and their families about the physiologic adaptation that can be expected to occur so that stigmatization for "failure" is minimized. As shown by abundant research, weight recovery is a natural trajectory following weight loss because patients biologically adapt to the loss of weight and fat mass, and patients need factual information to counterbalance the frustration of relapse. Improving care for obese patients requires NPs to fully inform patients about the reality of the after-after stage of weight loss.

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The authors and planners have disclosed that they have no financial relationships related to this article.

DOI-10.1097/01.NPR.0000512249.15712.a7

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